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EXAMINER	
NOBLE, MARCIA STEPHENS	
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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/642,272	HATTORI ET AL.	
	Examiner	Art Unit	
	Marcia S. Noble	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 November 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,7 and 15-35* is/are pending in the application.

4a) Of the above claim(s) *15-32* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1, 7, 33-35* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Continued Examination Under 37 CFR 1.114

1. A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 11/19/2007 has been entered.

Status of Claims

2. Claims 1, 7, and 15-35 are pending. Claims 1, 7, and 33-35 are amended, claims 8, 14, and 36-38 are canceled by the amendment to the claim set, filed 11/19/2007.

Election/Restrictions

3. Claims 15-32 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected subject matter, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in the reply filed on 6/13/2006. However, Applicant's traversal arguments were not found persuasive, and the restriction requirement was made final, as set forth in the office action mailed 8/9/2006 (page 2).

In the Restriction Requirement, mailed 1/13/2006, Applicant was required to make a species election for claim 7 for the genus, disease. Applicant elected chronic heart failure in their response, filed 6/13/2006. In the amendment to the claims, filed 11/19/2007, Applicant limited claim 7 to encompass chronic heart disease, ischemic heart disease, and ischemic heart failure. Because these conditions all fall within the genus, heart disease, therefore having overlapping scope, and because the amended claims encompass a gene therapy of the heart, ischemic heart disease and ischemic heart failure will be rejoined and considered with the elected species, chronic heart failure.

Claims 1, 7, and 33-35 are under consideration.

Claim Objections

4. Claims 1 and 7 are objected to because of the following informalities: Claims 1 and 7 recite, "decreased expression of AOP-1 gene". This is grammatically incorrect. An article, such as "an", should precede "AOP-1 gene". Appropriate correction is required.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Upon further consideration of the specification, the art, and amended claims, the scope of enablement rejection has been modified to encompass a full lack of enablement as follows:

5. Claims 1, 7, and 33-35, as amended and previously presented, are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

While determining whether a specification is enabling, one considers whether the claimed invention provides sufficient guidance to make or use the claimed invention, if not, whether an artisan would require undue experimentation to make and use the claimed invention and whether working examples have been provided. When determining whether a specification meets the enablement requirements, some of the factors that need to be analyzed are: the breadth of the claims, the nature of the invention, the state of the prior art, the level of one of ordinary skill, the level of predictability in the art, the amount of direction provided by the inventor, the existence of working examples, and whether the quantity of any necessary experimentation to make or use the invention based on the content of the disclosure is "undue".

Furthermore, USPTO does not have laboratory facilities to test if an invention will function as claimed when working examples are not disclosed in the specification, therefore, enablement issues are raised and discussed based on the state of knowledge

pertinent to an art at the time of invention, therefore skepticism raised in the enablement rejections are those raised in the art by artisans of expertise.

The instant claims are drawn to a method of treating a disease associated with decreased expression of an AOP-1 gene or AOP-1 comprising administering by direct injection or catheter-based delivery to heart cells of an individual an expression vector comprising a nucleic acid and a promoter wherein said nucleic acid enhances expression of AOP-1 and is (a) a nucleic acid encoding AOP-1 (claims 1 and 33-35) or (b) a nucleic acid that hybridizes under stringent conditions to a complementary strand of a nucleic acid encoding AOP-1 and encodes a polypeptide that retains the function of AOP-1 (claim 1). Narrowing embodiments specify that the disease be chronic heart disease, ischemic heart disease, or ischemic heart failure (claim 7).

The specification discloses the purpose of the instant invention is to provide methods of preventing or treating diseases associated with decreased expression of AOP-1 gene or protein (p. 6, [0011]). The specification teaches that AOP-1 also is called MER5 and peroxiredoxin 3 (p. 5, [0010]). The specification provides in vitro evidence that that cardiac myocytes transfected with AOP-1 are provided protection by AOP-1 against damage and loss of cell viability induced by hypoxia and reperfusion following hypoxia (p. 41-42, [00132]). Working example 16 also provides a method of delivering an adenoviral vector encoding an AOP-1 gene, operably linked to a CMV promoter and a poly A signal from bovine growth hormone (p. 40, [00127]), to the left ventricle of the heart via a catheter in a chronic heart disease rodent model (p. 48, [00151]). Four to five days following administration of the AOP-1 expression vector, the

heart and lungs were excised and prepared in a Langendorff perfusion system and subjected to ischemic perfusion experiments ex vivo. Cardiac ischemia was induced in the rat heart by stopping perfusion to the heart for 35 minutes and then resuming blood perfusion (p. 49, [00152]). AOP-1 gene delivery to the heart prior to ischemia resulted in significantly better functional recovery during reperfusion following induced ischemia in the heart. The time from ischemia to ischemic rigidity in the rat heart was significantly extended in the AOP-1 treated heart compared to sham controls. Also, LDH release by the heart, a marker correlated with cell injury and necrosis, was significantly repressed in the AOP-1 treated group compared to controls (p. 49, [00153]).

The instant claims encompass a method of treating a disease, more specifically chronic heart failure, ischemic heart failure, or ischemic heart disease, associated with decreased expression of an AOP-1 gene or AOP-1. The art teaches that mouse AOP-1, called MER5 was originally isolated from murine erythroleukemia cells and is involved in erythroid cell differentiation (Nemoto et al Gene 91(2):abstract, 1990). MER5 has also been suggested to have antioxidant properties in E. coli (Tsuji et al of record, page 377). However, the art does not teach an association between decreased expression of an AOP-1 gene or AOP-1 and chronic heart failure, ischemic heart failure, or ischemic heart disease. Therefore, an artisan would look to the specification for such teachings.

The specification teaches that rats were subjected to ischemic reperfusion experiments by the Langendorff's method following pretreatment with an AOP-1 expression vector (p. 48, [00151]). Langendorff's method is an ex vivo method that excises the heart and controls the perfusion of the heart. Therefore, this method can be

used to induce ischemia of the heart by stopping perfusion of the heart. It is an art accepted model for studying the biochemical and histological effects of ischemia on the heart (Skrzvpiec-Spring et al. J Pharmicol Toxicol Methods 2007 Mar-Apr;55(2):121-122, 2007; Sutherland and Hearse Pharmacol Res 41(6):626-627, 2000). However, while the Langendorff model is an accepted method for directly assessing the effects of ischemia on the heart, it is a supraphysiological experimental model that is devoid of its natural humeral and neurological influences and does not serve as a model for all ischemic conditions of the heart, such as ischemic arrhythmia and diseases of the heart (Skrzbpiec-Spring et al, p. 113, col 2, 117-118, and 120, col 2, last par). While the instant specification provides an experimental model that assesses ischemia in the rat heart following treatment with an AOP-1 expression vector, the disclosed method would not be considered a therapeutic method or a method for modeling or treating ischemic heart failure, chronic heart disease, or ischemic heart disease, as claimed, because the experimental model is a supraphysiological model that does not mimic the pathology of these diseases. Therefore, because the experiments taught by the specification do not teach a therapeutic model for chronic heart disease, ischemic heart failure, and ischemic heart disease, the instant specification does not teach an association between decreased expression of an AOP-1 gene or AOP-1 and chronic heart disease, ischemic heart disease, or ischemic heart failure.

Furthermore, for an artisan to use the instantly claimed therapeutic method to treat an individual, an artisan would have to apply the teaches of the specification to an animal model with ischemic heart failure, ischemic heart disease, or chronic heart

disease and determine if the delivery of an AOP-1 expression vector that enhanced AOP-1 expression would have a therapeutic effect in such an animal model. Then ultimately, if the animal models demonstrate a therapeutic effect, the method would have to be applied to humans with chronic heart disease, ischemic heart failure, or ischemic heart disease to determine if the method also has a therapeutic effect in human. This level of empirical experimentation to establish if the instant method would be therapeutic would be considered undue. Therefore, because the methods taught by the specification are not a therapeutic method and do not teach an association between decreased expression of an AOP-1 gene or AOP-1 and chronic heart disease, ischemic heart disease, or ischemic heart failure and because experimentation to determine if the instant method would be therapeutic is considered undue experimentation, the instant invention is not enabled as therapeutic method as claimed.

The amended claims encompass a therapeutic method for any disease associated with decreased expression of an AOP-1 gene or AOP-1 by delivering a therapeutic expression vector to the heart. Therefore, the instant claims encompass delivering a therapeutic expression vector to heart cells and treating a neurodegenerative brain disease, which would be considered an indirect administration of an expression vector.

The instant claims also encompass the use of a catheter-based delivery to heart cells. However, the breadth of a catheter-based delivery encompasses such limitations as catheterizing the femoral artery and delivering the expression vector systemically to the heart, which also would encompass an indirect route of administration.

However, gene therapies that utilize indirect routes of administration are shown to be highly unpredictable in the art as discussed in previous office actions. As set forth in the previous Office Actions, mailed 8/9/2006 (see bottom p. 12 to p. 13) and 5/17/2007 (p. 16), "Tomasoni and Benigni (Current Gene Therapy 4: 115, col 1 lines 4-7) state, "the success of gene therapy largely depends on an efficient delivery system for the transfer and expression of the therapeutic gene in the target organ or tissue." Many forms of vector delivery to a body site have been described in the art, but very few predictably deliver a therapeutic dose of a vector to the site of treatment. Gautam et al (Am J Respir Med, 1(1) abstract) discloses the use of different vector delivery routes to the lung, such as intravenous injection, intratracheal installation, and aerosol with varying degrees of success. They further disclose various barriers to delivery of vectors such as serum proteins during intravenous injection, surfactant and mucus interference during more topical applications of vectors. Systemic deliveries of some vectors have been demonstrated to be problematic because they induce immune and cytokine responses against the vector obstructing delivery of gene therapies (Gunther et al, Curr Med Chem – Anti-Cancer Agents, 5:p. 157, col 2, par 2, lines 14-17 to p. 158, col1 par 1, lines 1-3). Therefore, these problematic factors of immune response and alternative tropisms of the vectors preclude the delivery of these vectors by other means than direct administration to the target site.

In terms of specific delivery to the heart, Hajjar et al teaches, "...the vector must be delivered to the affected tissues. This poses a particularly formidable barrier in

conditions with an extensively distributed phenotype [p. 617, col 1, par 2, lines 11-14]...direct injection of adenovirus into the ventricular wall using an epicardial approach has also been shown to include significant expression of reporter constructs, however, the expression was focal, and the injections with the myocardium caused needle damage. Intramyocardial delivery of adenovirus using an intraventricular approach with retroinfusion of the coronary veins has also been used in larger animals yielding regional areas of transduction. In rodents, injection of an adenovirus carrying β-galactosidase into the pericardial sac transduced only the pericardial cell layers....[p. 617 bridging cols 1 and 2]." Overall, the art suggests that indirect administration is unpredicatable because various biological and physical barriers, such as an immune response to the vector, mucosal barriers, or focal perfusion, interfere with delivery of a therapeutic dose of an expression vector to the target cells.

Therefore, since the art teaches that gene therapies by indirect routes of administration are problematic and unpredictable, an artisan would look to the specification for guidance to overcome the unpredictabilities described in the art. However, the specification fails to teach means of overcoming the unpredictabilities of indirect administration of expression vectors. Therefore, an artisan would not know how to predictably do or use a therapeutic method to treat any disease other than a heart disease that encompasses direct delivery of an expression vector to heart cells because an indirect administration via heart cells to treat a non-heart related disease is unpredictable. Therefore, the specification only enables a therapeutic method for heart disease that comprises delivery of an expression vector to heart cells of an individual.

The breadth of the claims still encompasses introducing an expression vector comprising a nucleic acid that does not have operably linkage to a promoter. In the instant claims, the expression vector only requires that a promoter be present. They do not require that the promoter be functional or that the promoter drive expression of the nucleic acid present in the expression vector.

However, as discussed in the previous office actions, "for a gene therapy agent to be expressed effectively it must minimally comprise the elements to be directed by the transcription and translation machinery of the target cell which require a promoter capable of driving expression in the target cell. In the instant case, the specification discloses an adenoviral vector comprising the coding sequence for the AOP-1 gene operably linked to the CMV promoter and flanked on the 3' end of the AOP-1 gene a PolyA signal peptide. These elements resulted in the expression of AOP-1 in the heart when directly delivery to the heart....Because of the necessity for the minimal elements necessary to drive expression of a gene, an artisan would not know how to use a nucleic acid encoding AOP-1 without operable linkage to a promoter, in a gene therapy method that would result in the expression of the therapeutic gene." (See par bridging pages 14 and 15 of the Office Action, mailed 8/9/2006.)

Therefore, because the instantly claimed invention would require that the promoter of the expression vector be functional in heart cells and drive the expression of the nucleic acid present in the expression vector, the instant claims are only enabled for an expression vector comprising a nucleic acid operably linked to a promoter.

The instant claims encompass a nucleic acid that hybridizes under stringent conditions to a complementary strand of a nucleic acid encoding AOP-1 and encodes a polypeptide that retains the function of AOP-1. The breadth of this recitation encompasses any nucleic acid that non-specifically hybridizes to AOP-1 nucleotides under stringent conditions and have any analogous function to AOP-1.

However, the state of the art suggests that sequences identified by their hybridization properties are unpredictable in their identity in sequence to the original sequence to which it hybridized. Kennell teaches that 25 to 50% nucleic acid identity is all that is necessary for hybridization of a sequence under any conditions and that obtaining non-specific hybridization products are highly common in the art (par bridging p. 260 and 261 and par 1 of p. 261). The specification provides general guidelines and conditions for obtaining hybridization products. However, these conditions are exemplary and not limiting. Furthermore, these general guidelines and conditions provided by the specification do not provide any guidance to overcome the unpredictabilities described in the art. Therefore, an artisan would not know if a sequence that hybridized to AOP-1 would be a true complementary sequence capable of driving transcription or a non-specific hybridization product. Furthermore, for an artisan to use or make the claimed nucleic acid capable of hybridizing to the nucleotides of AOP-1, they would first have to sequence the product to determine if it was a true complement and then also test the functionality of the nucleotide for its promoter activity. This level of experimentation would be considered undue.

The limitation further requires that the complementary sequence that hybridizes to the nucleotide sequence encoding AOP-1 have the same function as AOP-1. However, as taught by Kennell et al and discussed above only 25 to 50% homology is required for hybridization under stringent conditions. Therefore, in the instant case, the limitation encompasses any sequence with 25 to 50% homology and that encodes a protein with analogous function as AOP-1. This could encompass a wide range of sequences. However, the specification does not teach such sequences encoding proteins with analogous function to AOP-1 nor does the specification provide teaching of hybridization conditions to obtain such proteins. Therefore, an artisan would not know how to use the instant method as claimed because it would not know how to predictably identify such functionally analogous proteins to AOP-1 as claimed.

In summary, because the specification does not teach a therapeutic method for treating a disease associated with decreased AOP-1 gene or protein expression but rather an ex vivo experimental model for induced ischemia of the heart, the instant therapeutic method is not enabled by the specification. Also, the art states that indirect routes of administering gene therapy vectors are unpredictable and the specification does not overcome these unpredictabilities. Therefore, the instant claims are not enabled for such gene therapies that encompass indirect administration as claimed. Also because the claims lack the required element of a promoter operably linked to a nucleic acid of the expression vector, the claims are not enabled for and expression vector a nucleic acid and a promoter without operable linkage as the claim encompasses. Lastly, because conditions of stringent hybridization still result in

unpredictable binding of non-specific sequences and the specification does not teach a means of overcoming these unpredictabilities, the instant claims are not enabled for a nucleic acid that hybridizes under stringent conditions to a complementary strand of a nucleic acid encoding AOP-1 and encodes a polypeptide that retains the function of AOP-1.

Applicant traverses the scope of enablement rejection as set forth in the previous office action, mailed 5/17/2007, on the following grounds:

1) The claims were deemed not to lack enablement for prophylactic methods, administration to any cell by any means (i.e.-indirect administration), and lacking operable linkage to a promoter (See last par on page 7 and first par on page 8 of remarks, filed 11/19/2007.).

Applicant asserts that the claims have been amended to recite "heart cells", "a promoter", and have removed reference to prophylactic methods. Therefore, the amendments to the claims obviate these issues of enablement.

Applicant's arguments have been considered and are not found persuasive for reasons discussed above. To reiterate, although the amended claims encompass delivery specifically to heart cells, the claims still encompass an indirect route of administration which is not enabled due to the art-disclosed unpredictabilities. Although the claims now recite, "a promoter", the full breadth of this recitation is not enabled because it encompasses any promoter including one that is non-functional in heart cells and one that is not operably linked to the nucleic acid of the expression vector.

Therefore, because the amendment to the claims do not fully address the issues of enablement, Applicant's arguments are not found persuasive and these grounds of rejection are maintained as previously made of record and as described above.

2) As a demonstration of the unpredictability of the art, the Office Action, mailed 8/9/2006, recited Chermille et al, which discussed the unpredictabilities of adenoviral vectors for use in human gene therapies (page 17).

Applicant traverses this ground of rejection on the grounds that several patents, whose applications were prosecuted before the filing of the instant application, demonstrate that viral vectors were enabled in the art for human gene therapy for heart disease (see par 3 on page 8 of remarks, filed 11/19/2007). Applicant refers to Pat No. 6,306,830 encompassing the use of viral vectors in a method of enhancing cardiac function in a human with congestive heart failure. Applicant also refers to Pat No. 6,436,908 encompassing the use of viral vectors in methods to improve myocardial function. Applicant also refers to Pat No. 6,589,523 encompassing the use of viral vectors in methods of treating a patient suffering from cardiomyopathy. These arts have been fully considered and Applicant's arguments are found persuasive. Therefore, this aspect of the enablement rejection is withdrawn.

3) The instant claims were only deemed enabled for a rat model as reduced to practice in the instant specification.

Applicant traversed this rejection on the grounds that many non-human animal models have been used and found useful for their applicability to humans. Applicant refers to Pat No. 6,306,830, Pat No. 6,436,908, and Pat No. 6,589,523 which teach

several cardiac gene therapies that are reduced to practice in various non-human mammals and whose finding are extended and applicable to humans (See pages 8 and 9 of remarks, filed 11/19/2007). Applicant also asserts that human AOP-1 expression by cells has a similar effect to that established for rat AOP-1 in rat cells and therefore human and rat AOP-1 have the same function. Therefore the results from the rat model disclosed in the specification are applicable and can be extrapolated to human (See pages 9 to 10 of remarks and Exhibits A and B, filed 11/19/2007).

As set forth in the remarks, "Applicants provide an article by Wonsey et al. that demonstrates the mitochondrial activating effect of human Prx-3 in R1A cells (fibroblast cell line) and MCF7 cells.... Figure 4 of Wonsey et al. shows cell distribution analysis conducted using NAO as an index of mitochondrial mass and DiOC6 as an index of mitochondrial membrane potential. This data indicates that forced expression of the Prx-3 gene elevates the mitochondrial membrane potential in these two types of cells [solid black line (control) vs. solid gray line (forced Prx-3 expression)].

Applicants have performed a similar analysis using cardiac myocytes. *See* Exhibit B.[3] In this experiment, Prx-3 showed a mitochondrial membrane potential activating effect when analyzed by two fluorescent indicators. Applicants submit that the data provided in Exhibit B demonstrates that the human Prx-3 has the same function as was discovered by Applicants using the rat Prx-3 gene."

The evidence of Wonsey et al and Exhibit B do suggest that both human and rat AOP-1 are serving analogous functions in mitochondrial membrane activation.

However, these arguments are not found persuasive because as discussed above in the modified enablement rejection. The specification does not provide adequate evidence for an association between decreased AOP-1 expression and a chronic heart disease, ischemic heart failure, or ischemic heart disease or a therapeutic method. Therefore, the instant invention is not enabled for treatment of an individual.

Claim Rejections - 35 USC § 112, 2nd Paragraph

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

6. The rejection of claims 33-38 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for reciting the limitation "said nucleic acid", which had insufficient antecedent basis, is withdrawn.

Applicant amended the claims to recite “said nucleic acid encoding AOP-1”, which now has sufficient antecedent basis. Therefore, the rejection is withdrawn.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

7. The rejection of claims 8 and 14, under 35 U.S.C. 102(b) as being anticipated by Tsuji et al. (1995; of record), is withdrawn.

Applicant canceled these claims rendering the rejection moot. Therefore, the rejection is withdrawn.

8. No claims are allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Marcia S. Noble whose telephone number is (571) 272-5545. The examiner can normally be reached on M-F 9 to 5:30.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Peter Paras can be reached on (571) 272-4517. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Marcia S. Noble

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